There are reports of NTM infections being contracted after tattooing[3,4] and acupuncture.[5] However, to our knowledge, this is the first reported case of *M fortuitum* infection associated with AET. Thus, practitioners should suspect mycobacterial infection in patients presenting with nodules and abscesses in areas that have been treated with AET. Such infections should be treated promptly to minimize further disfigurement.

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REFERENCES

1. Zhang XP, Jia CS, Wang JL, Shi J, Zhang X, Li XF, et al. Acupoint catgut-embedding therapy: superiorities and principles of application [article in Chinese]. Zhongguo Zhen Jiu 2012;32:947-51.
2. Lebrun-Vignes B, Kreft-Jais C, Castot A, Chosidow O. Comparative analysis of adverse drug reactions to tetracyclines: results of a French national survey and review of the literature. Br J Dermatol 2012;166:1333-41.
3. Mitchell CB, Isenstein A, Burkhart CN, Groben P, Morrell DS. Infection with *Mycobacterium immunogenum* following a tattoo. J Am Acad Dermatol 2011;64:e70-1.
4. Kappel S, Cotliar J. Inoculation of *Mycobacteria chelonae* from a tattoo. J Am Acad Dermatol 2011;64:998-9.
5. Guevara-Patino A, Sandoval de Mora M, Farreras A, Rivera-Olivero I, Fermin D, de Waard JH. Soft tissue infection due to *Mycobacterium fortuitum* following acupuncture: a case report and review of the literature. J Infect Dev Ctries 2010;4:521-5.

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Severe radiation dermatitis associated with concomitant vemurafenib therapy in a patient with metastatic melanoma

To the Editor: A 31-year-old woman was diagnosed with a nonulcerated 2.01-mm deep melanoma on her left arm. She underwent wide excision with negative surgical margins and a negative sentinel lymph node biopsy. Seven months later, she started having left hip pain that became progressively worse. A noncontrast magnetic resonance image revealed a large lesion in her left proximal femur that was subsequently biopsied, confirming the diagnosis of metastatic melanoma. Subcutaneous lesions on her trunk and thighs also developed. Biopsies revealed melanoma with positive BRAF V600E mutation. She was started on vemurafenib, a selective BRAF V600E inhibitor,[1] dosed at 960 mg twice daily. After 3 weeks of vemurafenib, she began receiving localized radiotherapy to treat her femoral lesion. After 1 week of daily radiotherapy, a brisk, raised, erythematous skin reaction restricted to her left anterior and posterior thigh developed. The erythematous regions were confined to the areas where she was receiving radiotherapy. With continuation of radiotherapy for 3 more days, an acute, rapidly worsening, and extremely painful burning sensation developed in her left anterior and posterior thigh. Physical examination revealed blistered and erythematous skin with dry and moist desquamation in the radiotherapy fields on her left anterior and posterior thigh, indicative of a worsening skin reaction consistent with radiation burns. She had received a total radiation dose of 30 Gy in 10 uninterrupted daily 3-Gy fractions as originally planned. With discontinuation of radiotherapy, her radiation dermatitis healed after 1 month.

Two cases of localized radiation dermatitis that resolved with topical corticosteroids and did not require cessation of vemurafenib have been reported in patients who started vemurafenib after completion of radiotherapy.[2] Another case of radiodermatitis was reported in a patient receiving concomitant vemurafenib and radiotherapy who had been earlier administered 60 Gy of local radiotherapy to the same area that 4.5 years later developed radiodermatitis upon administration of 20 Gy of radiotherapy.[3] To our knowledge, our case represents the first reported instance in the English literature of localized radiation dermatitis developing in a patient receiving concomitant vemurafenib and radiotherapy who was previously naive to both treatment modalities. Sustained erythema seen in radiation dermatitis typically manifests 10 to 14 days after dosing, whereas sustained erythema developed in our patient after 7 days of radiotherapy. Moreover, the severity of the radiation dermatitis seen in our patient after 10 days of radiotherapy is not usually seen until after 4 to 5 weeks of radiotherapy at doses of 40 Gy or greater. The radiosensitizing effect of vemurafenib leads to increased cellular damage and impaired DNA repair.[4] One proposed mechanism for radiosensitization secondary to vemurafenib suggests that vemurafenib activates wild-type BRAF in keratinocytes and leads to radiosensitization as a result.[3] However, the exact mechanism remains unclear. Dermatologists should

be aware that radiosensitization may be associated with vemurafenib administration and carefully monitor patients receiving concomitant radiotherapy, as well as patients who start vemurafenib after radiotherapy. Neither radiotherapy nor vemurafenib need be stopped if radiation dermatitis develops, in that reported cases resolved without further adverse events after topical corticosteroid administration, cessation of radiotherapy, or both.

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REFERENCES

1. Dummer R, Rinderknecht J, Goldinger SM. Ultraviolet A and photosensitivity during vemurafenib therapy. N Engl J Med 2012;366:480-1.
2. Boussemart L, Boivin C, Claveau J, Tao YG, Tomasic G, Routier E, et al. Vemurafenib and radiosensitization. JAMA Dermatol 2013; 149:855-7.
3. Satzger I, Degen A, Asper H, Kapp A, Hauschild A, Gutzmer R. Serious skin toxicity with the combination of BRAF inhibitors and radiotherapy. J Clin Oncol 2013;31:e220-2.
4. Hymes SR, Strom EA, Fife C. Radiation dermatitis: clinical presentation, pathophysiology, and treatment 2006. J Am Acad Dermatol 2006;54:28-46.

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Treatment of epidermolysis bullosa pruriginosa using systemic and topical agents

To the Editor: A 46-year-old Hispanic woman presented to our dermatology clinic with a 6-year history of an intermittent pruritic vesicobullous eruption on her lower legs and, to a lesser extent, on her forearms (Fig 1, *A*). She had failed to respond to prior treatments, including topical and intralesional corticosteroids, mycophenolate mofetil, and phototherapy. There was no family history of a similar condition. Histologic findings showed subepidermal clefts with minimal inflammation. Direct immunofluorescence on perilesional skin was negative. A complete workup for immunobullous disease and porphyria was negative. Electron microscopy demonstrated separation of the sublamina densa with a reduced number of anchoring fibrils, which was clinically consistent with dystrophic epidermolysis bullosa pruriginosa (EBP). DNA extraction from peripheral blood followed by bidirectional *COL7A1* gene sequencing (GeneDx, Gaithersburg, MD) revealed a novel compound heterozygotic mutation; a guanine (G) to cytosine (C) splice site mutation at the beginning of intron 51 (IVS51+1 G→C) and adenine (A) to thymine (T) resulting in a codon mutation of glutamine (Gln) to leucine (Leu) at position 1924 (p.Gln1924Leu) on exon 69.

Pruritus and excoriation are critical in unmasking skin fragility in EBP. Given the pauci-inflammatory nature of EBP, we focused on the control of pruritus with the use of ketamine 0.5% and amitriptyline 2% (KA) topical gel. With regular use of KA gel, the patient described a 90% decrease in localized pruritus and blistering. Subsequently, the patient was started on sertraline for depressive symptoms. Over the 6-month period on oral sertraline and KA gel, the patient had virtually complete resolution of localized and generalized pruritus with minor relapses (Fig 1, *B*).

Effective treatment of EBP is challenging, and long-term outcomes are rarely described in the literature. Treatments reported to date include corticosteroids, antihistamines, cryotherapy, tacrolimus, cyclosporine, and thalidomide.[1] However, control of inflammation has not demonstrated consistent efficacy. Based on the structural fragility of EBP, unmasked by severe pruritus and scratching, we advocate focusing the treatment on the control of pruritus, minimizing trauma, and managing lower extremity edema. Topical KA is thought to act pre- and post-axonally in the transmission of pain and possibly pruritus. Poterucha et al[2] showed that topical KA had a 62% response rate in a wide variety of cutaneous disorders, without inducing systemic side effects.[2,3] Interestingly, the concomitant use of sertraline for mild depressive symptoms further improved the patient's generalized pruritus and EBP. Sertraline has been reported as a first-line treatment for cholestatic pruritus and implicates serotonergic mediators in the pruritus of EBP.[4] The exact mechanism of action is unknown but is thought to have a regulatory action on the transmission of itch signals.[5]

The efficacy and minimal medication risk with the combined oral and topical therapies reported here favor this approach as a primary treatment of EBP, a disease that necessitates long-term management. Additional studies are required to further validate the efficacy of systemic sertraline and topical KA for treatment of EBP and possibly other skin conditions in which pruritus plays a key pathogenic role.